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TOWNSEND AND TOWNSEND AND CREW, LLP
TWO EMBARCADERO CENTER
EIGHTH FLOOR
SAN FRANCISCO, CA 94111-3834

EXAMINER	
LIU, SUE XU	

ART UNIT	PAPER NUMBER
1639	

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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	09/694,758	CHAKRAVARTI, SHUKTI	
	Examiner	Art Unit	
	SUE LIU	1639	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *12 June 2009*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *42 and 46-52* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *42 and 46-52* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *2/19/08*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Claim Status

1. Claims 1-41, 43-45, 53, 55 and 56 have been canceled as filed on 5/16/08.

Claims 42 and 46-52 are currently pending

Claims 42 and 46-52 are being examined in this application.

Election/Restrictions

2. Applicant's election of Group IV (original Claims 5-7) as previously acknowledged.

Priority

3. This application claims priority to U.S. Provisional Patent Application No. 60/160,835, filed 10/21/1999.

4. Applicant's claim for the benefit of a prior-filed application under 35 U.S.C. 119(e) or under 35 U.S.C. 120, 121, or 365(c) is acknowledged. Applicant has not complied with one or more conditions for receiving the benefit of an earlier filing date under 35 U.S.C. 119(e) or under 35 U.S.C. 120, 121, or 365(c) as follows:

The later-filed application must be an application for a patent for an invention which is also disclosed in the prior application (the parent or original nonprovisional application or provisional application). The disclosure of the invention in the parent application and in the later-filed application must be sufficient to comply with the requirements of the first paragraph of 35

U.S.C. 112. See *Transco Products, Inc. v. Performance Contracting, Inc.*, 38 F.3d 551, 32 USPQ2d 1077 (Fed. Cir. 1994).

The disclosure of the prior-filed application, Application No. 60, fails to provide adequate support or enablement in the manner provided by the first paragraph of 35 U.S.C. 112 for one or more claims of this application.

The instant claims recite methods of diagnosing ulcertative colitis (UC) or "Crohn's disease (CD) by determining at least a combination of five genes including "GRO3", "HNL", "MMP-12", "elafin", and "COL6A3". The said provisional application, 60/160,835, does not provide support for the said method of UC or CD diagnosis using the "combination" of specific five genes. The said provisional application only provides a list of various genes that were differentially expressed in different cell types. The said provisional application does not specifically recite any specific "combination" of genes that can be used as markers for the purposes of diagnosis of any particular disease.

Thus, the instant application does not obtain the priority benefit of the provisional application. The effective filing date of the instant claims (42, 45-52 and 54) is 10/23/2000.

Information Disclosure Statement

5. The information disclosure statement filed 2/19/08 has been considered. See the attached PTO-1449.

Claim Objection(s) / Rejection(s) Withdrawn

6. In light of applicants' amendment to the claims to recite "any of said … gene products" and "IBD phenotypes", the following claim rejections as set forth in the previous office action are withdrawn:

A.) Claims 42, 45-52 and 54 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement.

B.) Claims 42, 45-52 and 54 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement.

C.) Claims 42, 45-52 and 54 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement.

New Claim Objection(s) / Rejection(s)

Claim Rejections - 35 USC § 103

7. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

Alexander and Others

8. Claims 42 and 46-52 are rejected under 35 U.S.C. 103(a) as being unpatentable over **Alexander** et al (Digestive Diseases and Sciences, Vol. 41, No. 4 (April 1996), pp. 660-669; previously cited), **Dieckgraefe** et al. (Gastroeterology, vol 114, No. 4, April 1998; G3954; cited

previously), **Poulakkainen** (Gastroenterology. 114:A1064; 1998; previously cited), **Tekamp-Olson et al.**, (GenBank accession number X53800; 1995; cited in IDS), **Bartsch et al.**, (GenBank accession number S75256; 1995; cited in IDS), **Sallenave et al.**, (GenBank accession number L10343; 1993; cited in IDS) and **Chu et al.**, (GenBank accession number X52022; 1998; cited in IDS). This rejection is necessitated by applicant's amendments to the claims.

The instant claims recite "a method for determining whether a test colon cell has an inflammatory bowel disease (IBD) phenotype, said method comprising:

(a) determining an expression level of each of the following gene products in said test colon cell: (i) a macrophage inflammatory protein-23 (GRO3) gene product (SEQ ID NO:7); (ii) a neutrophil lipocalin (HNL) gene product (SEQ ID NO: 14); (iii) a macrophage elastase (MMP-12) gene product (SEQ 1I) NO:81); (iv) an elastase specific inhibitor (elafin) gene product (SEQ 133 NO:85); and (v) a type VI collagen α3 chain (COL6A3) gene product (SEQ ID NO:87);

(b) comparing the expression level of each of said GRO3, HNL, MMP-12, elafin, and COL6A3 genes gene products in said test colon cell to an expression level of the same gene product in a normal colon cell; and

(c) associating an increase in the expression level of any of said GRO3, HNL, MMP-12, elafin, and COL6A3 genes gene products in said test colon cell relative to the expression level of the'same gene product in said normal colon cell with an IBD phenotype in said test colon cell."

Alexander et al, throughout the publication, disclose a method to determine altered expression of protooncogenes (cell cycle related genes) in patients with inflammatory bowel disease (IBD), which reads on the determining gene expression of **clm 42**. The reference assayed transcripts of 15 protooncogenes (refer to IBD genes) in colonic epithelial cells of IBD patients

and controls (e.g., see abstract). The reference discloses that increased levels (refers to the differential expression of the instant claim) of soluble mediators (e.g. Leukotrienes, prostaglandins) of inflammation as well as the cells of immune system have been found to be present in the intestinal mucosa and submucosa of IBD patients (e.g., see page 660, last paragraph bridging first paragraph in page 661). The reference discloses expression of transcripts of eight growth factor receptor related genes in colonic epithelial cells of IBD patients and controls (i.e., see left column in page 661). These read on the comparison step of **clm 42**.

The reference also teaches cells obtained from patients with inflammatory bowel disease (Abstract of the reference), which reads on test colon cells of **clm 42**.

The reference also teaches samples are obtained from surgery (p. 661, right col., para 2), which reads on the sample of **clm 47**.

The reference also teaches hybridization analysis (e.g. northern blotting) to analyze gene expression (p. 662, right col.), which reads on the method step of **clm 48**.

The northern blotting membrane also reads on an array having a substrate, because the northern blotting membrane has probes bound thereto and the probes are arranged in a two dimensional matrix format (see Figure 2 of the reference).

Alexander et al do not explicitly teach the listed genes in the instant **clms 42** and **46**. The reference also does not explicitly teach using any array having 12-40 nucleotides in length as recited in **clms 49-52**.

However, the genes (such MMP-12) in listed in the instant Claim 42 (and Table 1 of the instant specification) are not novel genes, and are well known for their role in IBD. The specification in page 19, discloses 'Table 1 indicates those sequences which are over- or

underexpressed in a CD- or UC-derived cells relative to normal tissue.' Applicants in the specification disclose the GenBank accession numbers of the genes used in the claimed method. Thus, all the genes used in the claimed method are well known in the art.

In addition, **Dieckgraefe** et al, throughout the publication, disclose a method for identifying gene expressed in IBD. The reference has used GeneChip expression monitoring system to examine mucosal gene expression in ulcerative colitis, Crohns' disease, and both in inflamed and non-inflamed non IBD specimens (Background section of the reference). The reference also teaches RNA isolated from the mucosa of colonic resection specimens was used to generate hybridization probes (See Methods). The reference also teaches light directed solid-phase combinatorial chemistry was used to generate oligonucleotide probe array (see Methods), which reads on the nucleic acid probes, array, and substrates of **clms 48-52**. The reference teaches using GeneChip system (from Affymetrix), which represents nearly 7000 human cDAN and EST sequences, which inherently would encompass the five human genes listed in the instant claim 42, as evidenced by **Lawrance** et al. (Human Molecular Genetics. Vol.10(5): 445-456; 2001; cited in IDS; see especially page 454, right col., "Affymetrix… array set contained GenBank Gene and ESTs deposited in the database prior to 1997").

Dieckgraefe et al also teach the need to identify gene markers differentially expressed in IBD, and the need to use different genes that are differentially expressed to identify genotypes for the different diseases (such as CD and UC) for potential pharmaceutical purposes (see Aims section of the reference). The reference further teaches host defense molecules are over expressed in IBD cells (Results section).

Alternative, as discussed supra, all of the genes listed in **clm 42** are known genes and their sequences are available in the public in GenBank (as evidenced by the instant disclosed GenBank access numbers).

For examples, **Puolakkainen** et al (G4358), throughout the publication, teach distinct expression profiles of stromelysin-s, collagenase and **MMP-12** in intestinal ulcerations. As taught by Alexander et al, Crohn's disease (CD), and ulcerative colitis (UC) are part of larger group of IBDs (p. 660 of Alexander). The Puolakkainen reference also teaches the MMP-12 gene is "abundantly expressed" in test tissues when compared to normal tissues, which "abundant expression" read on the inherent property of the MMP-12 gene differential expression pattern (with at least a factor of two increase in expression).

Tekamp-Olson et al., (GenBank accession number X53800; 1995; cited in IDS), teaches the gene sequence or the gene for GRO3.

Bartsch et al., (GenBank accession number S75256; 1995; cited in IDS), teaches the gene sequence or the gene for HNL.

Sallenave et al., (GenBank accession number L10343; 1993; cited in IDS), teaches the gene sequence or the gene for Elafin.

Chu et al., (GenBank accession number X52022; 1998; cited in IDS), teaches the gene sequence or the gene for COL6A3.

Thus, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to measure all known gene (or to use microarrays comprising known

genes including MMP-12, GRO3, HNL, etc.) products expression in colon test cells, and to correlate gene expression pattern with IBD colon cell types.

A person of ordinary skill in the art would have been motivated to use all the known genes or include all known gene probes in an array as human gene arrays are commercially available. In addition, Alexander et al teach the need to determine differential gene expression in colon cells to study IBD (e.g. Abstract). The Dieckgraefe reference also teach the need to identify gene markers differentially expressed in IBD, and the need to use different genes that are differentially expressed to identify genotypes for the different diseases (such as CD and UC) for potential pharmaceutical purposes (see Aims section of the reference). As the Dieckgraefe reference teaches designing a microarray with various probes to represent various known human genes, and the various claimed human genes are known (as provided by the cited GenBank accession number), it would have been prima facie obvious for one of ordinary skill in the art to device an array including all known genes and to determine the expression profile of these genes in cells of interests (as this is the basis for microarray technology).

A person of ordinary skill in the art would have been motivated to use probes with specific length (such as 25-mer) to detect gene expression, because probes with different lengths are known in the art and they can be used to represent diverse genes, as taught by Dieckgraefe et al. It would have been obvious to one of ordinary skill in the art to apply the standard technique of tagging generating an array with probes of desired length taught by Dieckgraefe, to improve the DNA microarray for the predicable result of enabling standard gene expression profiling using DNA microarray.

A person of ordinary skill in the art would have reasonable expectation of success of achieving such modifications because the techniques for monitoring gene expression such as using DNA microarray and the specific genes (such as MMP-12) are known in the prior art as taught by the cited references, who have demonstrated the detection of expression of various genes in IBD cells.

Dieckgraefe, Poulakkainen, etc.

9. Claims 42 and 46-52 are rejected under 35 U.S.C. 103(a) as being unpatentable over **Dieckgraefe** et al (Gastrocterology, vol 114, No. 4, April 1998; cited previously), **Poulakkainen** (G4358; cited previously), and if necessary, in view of **Tekamp-Olson et al.**, (GenBank accession number X53800; 1995; cited in IDS), **Bartsch et al.**, (GenBank accession number S75256; 1995; cited in IDS), **Sallenave et al.**, (GenBank accession number L10343; 1993; cited in IDS) and **Chu et al.**, (GenBank accession number X52022; 1998; cited in IDS). This rejection is necessitated by applicant's amendments to the claims.

Dieckgraefe et al, throughout the publication, disclose a method for identifying gene expressed in IBD, which reads on the determining gene expression of **clm 42**. The reference has used GeneChip expression monitoring system to examine mucosal gene expression in ulcerative colitis, Crohns' disease, and both in inflamed and non-inflamed non IBD specimens (Background section of the reference), which reads on IBD of **clm 42**. The reference also teaches RNA isolated from the mucosa of colonic resection specimens was used to generate hybridization probes (See Methods), which reads on the surgical resection sample of **clm 47**. The reference also teaches light directed solid-phase combinatorial chemistry was used to generate

oligonucleotide probe array (see Methods), which reads on the nucleic acid probes, array, and substrates of **clms 48-52**. The reference also teaches dramatic changes of gene expression for a wide range of genes (Results section of the reference), which reads on the at least a factor of two difference in expression of **clm 46**.

Dieckgraefe et al also teach the need to identify gene markers differentially expressed in CD and UC, and the need to use different genes that are differentially expressed to identify genotypes for the different diseases (such as CD and UC) for potential pharmaceutical purposes (see Aims section of the reference). The reference further teaches host defense molecules are over expressed in IBD cells (Results section).

Dieckgraefe et al do not explicitly teach monitoring expressions of the specific listed genes in **clms 42** and **46**.

However, the genes shown in Table 1 (which comprises MMP-12) of the instant specification are publicly known and available. Furthermore, **Puolakkainen** et al, throughout the publication, teach distinct expression profiles of stromelysin-s, collagenase and MMP-12 in intestinal ulcerations. The Puolakkainen reference also teaches the MMP-12 gene is "abundantly expressed" in test tissues when compared to normal tissues, which "abundant expression" read on the inherent property of the MMP-12 gene differential expression pattern (with at least a factor of two increase in expression).

In addition, **Tekamp-Olson et al**., (GenBank accession number X53800; 1995; cited in IDS), teaches the gene sequence or the gene for GRO3.

Bartsch et al., (GenBank accession number S75256; 1995; cited in IDS), teaches the gene sequence or the gene for HNL.

Sallenave et al., (GenBank accession number L10343; 1993; cited in IDS), teaches the gene sequence or the gene for Elafin.

Chu et al., (GenBank accession number X52022; 1998; cited in IDS), teaches the gene sequence or the gene for COL6A3.

The Dieckgraefe reference teaches using GeneChip system (from Affymetrix), which represents nearly 7000 human cDAN and EST sequences, which inherently would encompass various human genes.

Thus, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to measure all known gene (or to use microarrays comprising known genes including MMP-12, GRO3, HNL, etc.) products expression in colon test cells, and to correlate gene expression pattern with IBD colon cell types.

A person of ordinary skill in the art would have been motivated to use all the known genes or include all known gene probes in an array as human gene arrays are commercially available. In addition, the Dieckgraefe reference teaches the need to identify gene markers differentially expressed in IBD, and the need to use different genes that are differentially expressed to identify genotypes for the different diseases (such as CD and UC) for potential pharmaceutical purposes (see Aims section of the reference). As the Dieckgraefe reference teaches designing a microarray with various probes to represent various known human genes, and the various claimed human genes are known (as provided by the cited GenBank accession number), it would have been prima facie obvious for one of ordinary skill in the art to device an

array including all known genes and to determine the expression profile of these genes in cells of interests (as this is the basis for microarray technology).

A person of ordinary skill in the art would have reasonable expectation of success of achieving such modifications because the techniques for monitoring gene expression such as using DNA microarray and the specific genes (such as MMP-12) are known in the prior art such as taught by Dieckgraefe et al and Puolakkainen et al, who have demonstrated the detection of expression of various genes in IBD cells.

Cock and others

10. Claims 42 and 46-52 are rejected under 35 U.S.C. 103(a) as being unpatentable over **Cocks** et al (US 6,607,879; 08/19/2003; Filed on 2/9/1998; cited previously), in view of **Dieckgraefe** et al (Gastroeterology, vol 114, No. 4, April 1998; cited previously), and if necessary, in view of **Bartsch et al**., (GenBank accession number S75256; 1995; cited in IDS), **Sallenave et al**., (GenBank accession number L10343; 1993; cited in IDS), **Chu et al**., (GenBank accession number X52022; 1998; cited in IDS), and **Shapiro** et al. (GenBank accession number L23808; 1994; cited in IDS). This rejection is necessitated by applicant's amendments to the claims.

Cocks et al, throughout the patent, teach methods of diagnosing or monitoring diseases such as Crohn's diseases, and ulcerative colitis (IBDs) using DNA microarray (Abstract and Claims 4-5 of the reference), which reads on the IBDs of **clm 42**, and the array method of **clm 48**. The reference also teaches that SEQ ID No 1100 is human cytokine (GRO-γ) (See Table 1 of the reference), which reads on GRO3 of **clms 42** and **46**.

The reference further teaches that the transcripts (mRNA) used with the array are obtained from various sources such as inflamed samples and noninflamed biological samples from various tissues such as hematopoietic tissues or colon tissues (Col. 7, 1st paragraph and lines 10-25), which would read on gene product from a test cell and a control cell (step b) of **clm 42**, and the tissue sample of **clm 47**. In addition, the reference teaches comparing the hybridization pattern from diseased and non-diseased samples (Claim 4), which reads on steps (b)-(c) of **clm 42**.

The reference teaches cDNAs of various genes are immobilized on a substrate and are hybridizable elements on a microarray (Claims 2 and 3 of the reference), which reads on nucleic acid probes that specifically hybridize to the gene product of **clm 49**. The reference further teaches that transcript levels are preferably at least about 2xhigher in a diseased sample than in the nondiseased sample (Col. 7, lines 22-25), which reads on the expression level of **clm 46**. Furthermore, the reference teaches that the polynucleotide probes can be synthesized on the surface of the substrate by using covalent bonding to the substrate (Col. 10, lines 20-22, for example), and the substrates used could be chips, membrane, plates, etc. (Col. 10, lines 1-5), which read on the array and its substrate of **clms 49-52**.

Cocks et al do not explicitly teach using probes with lengths ranging from 12-40 nucleotides, as recited in **clm 49**. The reference also does not explicitly teach monitoring expressions of the specific listed genes in **clm 42**.

However, Dieckgraefe et al (G4358), throughout the publication, teach using probes with length of 25 nucleotides. The reference also teaches using GeneChip system (from Affymetrix), which represents nearly 7000 human cDAN and EST sequences, which inherently would

encompass various human genes. Dieckgraefe et al also teach the need to identify gene markers differentially expressed in CD and UC, and the need to use different genes that are differentially expressed to identify genotypes for the different diseases (such as CD and UC) for potential pharmaceutical purposes (see Aims section of the reference). The reference further teaches host defense molecules are over expressed in IBD cells (Results section).

In addition, **Bartsch et al**., (GenBank accession number S75256; 1995; cited in IDS), teaches the gene sequence or the gene for HNL.

Sallenave et al., (GenBank accession number L10343; 1993; cited in IDS), teaches the gene sequence or the gene for Elafin.

Chu et al., (GenBank accession number X52022; 1998; cited in IDS), teaches the gene sequence or the gene for COL6A3.

Shapiro et al. (GenBank accession number L23808; 1994; cited in IDS), teaches the gene sequence or the gene for MMP-12.

Thus, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to use probes with specific length to detect gene expression product, and to use the expression profile to distinguish between UC and CD.

Thus, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to measure all known gene (or to use microarrays comprising known genes including MMP-12, GRO3, HNL, etc.) products expression in colon test cells, and to correlate gene expression pattern with IBD colon cell types, as well as using probes with the desired length.

A person of ordinary skill in the art would have been motivated to use all the known genes or include all known gene probes in an array as human gene arrays are commercially available. In addition, the Dieckgraefe reference teaches the need to identify gene markers differentially expressed in IBD, and the need to use different genes that are differentially expressed to identify genotypes for the different diseases (such as CD and UC) for potential pharmaceutical purposes (see Aims section of the reference). As the Dieckgraefe reference teaches designing a microarray with various probes to represent various known human genes, and the various claimed human genes are known (as provided by the cited GenBank accession number), it would have been prima facie obvious for one of ordinary skill in the art to device an array including all known genes and to determine the expression profile of these genes in cells of interests (as this is the basis for microarray technology).

A person of ordinary skill in the art would have been motivated to use probes with specific length (such as 25-mer) to detect gene expression, because probes with different lengths are known in the art and they can be used to represent diverse genes, as taught by Dieckgraefe et al. It would have been obvious to one of ordinary skill in the art to apply the standard technique of tagging generating an array with probes of desired length taught by Dieckgraefe, to improve the DNA microarray for the predicable result of enabling standard gene expression profiling using DNA microarray.

A person of ordinary skill in the art would have reasonable expectation of success of achieving such modifications because the techniques for generating probes with certain length is known in the art, as evidenced by Dieckgraefe et al.

Conclusion

Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Sue Liu whose telephone number is 571-272-5539. The examiner can normally be reached on M-F 9am-3pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Christopher Low can be reached at 571-272-0951. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

/SUE LIU/
Primary Examiner, Art Unit 1639
8/28/09